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A CONTRIBUTION TO THE CLINICAL HISTORY OF NON-SUPPURATIVE, ACUTE (HÆMORRHAGIC) ENCEPHALITIS, WITH THE REPORT OF A CASE FOLLOWING AN ATTACK OF MUMPS.

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THE subject of the non-suppurative acute encephalitis has been handled so recently and in so masterly a manner by Oppenheim,[1] that I should hesitate to bring it up again, were it not that in this country but few cases seem to have been observed, and that the clinical interest of the disease is distinctly great.

My personal interest in the matter began with the observation of what I believe to have been a typical case of this sort, during the epidemic of influenza in 1892. This case was reported in a paper on some of the nervous complications of influenza.[2] The doubt which existed in my mind as to the diagnosis, based on the fact that several weeks had elapsed between the disappearance of the influenza and the onset of the cerebral symptoms, would now be less than it was then, because, as I shall point out, the presence of this interval seems to be rather characteristic.

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[1] *Deutsche Zeitschr. für Nervenheilkunde.* 1895, vol. 6, p. 375.

[2] *Boston Medical and Surgical Journal,* October 6, 1892.

Since seeing this case I have met with another of still greater importance, which I shall report in the present paper. The practical interest attaching to my cases is that they confirm the important conclusion reached by Oppenheim; namely, that in spite of the extremely serious character of the symptoms, and although they indicate the simultaneous involvement of a number of important centres, complete or partial recovery is by no means uncommon.

The forms of encephalitis which are to be considered in this connection are those in which multiple foci of inflammation are met with, of variable extent, and characterized by the presence of signs of congestion, leucocyte infiltration, minute and even massive hæmorrhages, with greater or less destruction of nerve elements, but not by suppuration.[3] The same cause which produces the encephalitis, such as the influenza bacillus, may excite also meningitis, but when the meningitis becomes a prominent feature, as in the cerebro-spinal form, a new element of danger is introduced. It is also obvious that if a complete discussion of the subject from a pathological standpoint was to be attempted, other infectious diseases of the nervous system, such as disseminated myelitis and neuritis multiplex, would have to be considered.

The principal, though by no means the only, clinical symptom groups which deserve attention in connection with the subject of this paper are, (1) Wernicke's polio-encephalitis superior, in which the gray matter of the third ventricle, of the aqueduct of Sylvius, and of the fourth ventricle is chiefly involved; and (2) the form which was first distinctly pointed out by Strümpell[4] in 1890, where the lesions involve more especially the hemispheres, especially the cortex and the basal ganglia, or the cerebellum, and often both sides in a symmetrical fashion, causing a variety of symptoms, such as convulsions, aphasia, monoplegias and hemiplegias. Optic neuritis may be met with, and severe general symptoms, fever and coma, are always present. Even the ocular paralyses may be present, in connection with other symptoms, and thus the cases of this group touch hands with those of the other.

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[3] It is to be borne in mind that, in view of the present inadequate state of our knowledge with regard to the anatomical changes present in the slighter cases of this class, and, on the other hand, in the light of our information with regard to the effects of bacterial and other toxines in general, we can by no means be sure but that some of the symptoms attributed to local inflammation are really due purely to toxine poisoning.

[4] In view of the early and important contributions of Leichtenstern to this subject Oppenheim designates these cases as of the Strümpell-Leichtenstern type. (See *Deutsche Med. Wochenschr.* 1890 and 1892).

The Wernicke symptom-complex differs from that of the Strümpell form of encephalitis in that paralysis of the external muscles of the eye is present in a high degree, and that optic neuritis is of more common occurrence. The motion of the limbs may be impaired, and the speech affected, and general symptoms similar to those observed in the other form are likely to show themselves. The temperature may be elevated, or normal, or subnormal. The genesis of these diffuse cerebral symptoms has recently been studied anew by Jacobaeus,[5] who thinks that alcoholic neuritis with its cerebral and spinal complications plays an important part in their productions.

Most of the cases of this class certainly affect alcoholic subjects, and in them the outcome has almost always been fatal, though a few cases of other origin have ended in recovery. (See Jacobaeus, l. c.)

The clinical and experimental evidence is strong that these focal myelitic and encephalitic processes may be due to bacterial toxines of various sorts, and that the outbreak of encephalitis or myelitis may either attend or follow the primary manifestations of the infection, or may itself be the primary manifestation. A number of facts bearing on these points were cited in a summary of the results of infectious diseases, published by me in 1895)[6], and several new observations have appeared within the past year.

So far as human pathology is concerned, the poison of influenza appears to be the most active of the toxic causes of acute lesions, and the influenza bacillus has been found in the diseased foci, but the case to be reported to-day is especially interesting, because it seems to show that the mumps may be responsible for equally serious results. It should not, of course, be forgotten in this connection, that the real agent may be a secondary infection. Attention has indeed been called to the especial need of caution in this respect in the case of affections following the mumps, because there are some grounds for suspecting that the organism which causes this disease may not be, strictly speaking, bacterial, but rather protozoal.

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[5] Ueber einen Fall von Polioencephalitis hæmorrh.sup *Deutsche Zeitschr. f. Nervenheilkunde*, 1895, p. 334. (See also Kaiser, cited below).

[6] Relation of Infectious Processes to Diseases of the Nervous System. *Am. Journal of Med. Sciences*, 1895, Vol. 109, p. 254.

The cases of the form of encephalitis hitherto described by Strümpell, Leichtenstern, Fürbringer, and others, are, as Oppenheim justly observes, not well calculated to give a correct idea of the prognosis of the disease in question, because they were selected mainly in the interests of the pathological anatomy. A number of recoveries are, however, to be found among them.

Oppenheim himself adds six new cases, which in brief outline are as follows:

The first case was that of a girl of sixteen; the whole duration four months; the outcome favorable. The main symptoms were headache; motor and sensory aphasia, ushered in by speechlessness; optic neuritis. There was but trifling elevation of temperature.

The second case was that of a girl of only ten years; the duration of the illness about two months; the outcome favorable. The onset occurred three months after a slight attack of influenza, which may or may not have been responsible for the result. The symptoms were vertigo and prostration, passing rapidly into unconsciousness lasting three days; fever of short duration; loss speech; stiffness of neck muscles; slight optic neuritis.

The third case was that of a woman of twenty eight. The duration of the illness, including a prolonged convalescence, was eight months. The onset was moderately rapid, and the first symptoms showed themselves in less than a month after an attack of influenz. The symptoms were, mental confusion; loss of speech; paresis of right arm and side of face, preceded by an epileptic seizure.

The fourth case was, again, that of a young girl of twelve. The duration of the illness was five or six months; the outcome unfavorable, and the cause unknown. The symptoms were paresis of the limbs and face, with partial degenerative reaction of the left facial muscles, ataxia, impairment of sense of position of the limbs, exaggerated knee jerk and ankle clonus, impairment of hearing, paresis of the palate.

The fifth case was that of a man of twenty. The duration of the illness was four months, but the recovery incomplete. Here also there were widespread sensory and motor symptoms, disturbed articulation, hemianopsia, double optic neuritis, tinnitus aurium.

The sixth case, that of a man of twenty-seven, is of especial interest, since it resembles that of my patient in the respects that complete ophthalmoplegia and difficulty in deglutition were present, associated with motor disorder of the face and limbs, and impaired deglutition.

Since the publication of Oppenheim's paper some further contributions of importance have been made to both the clinical and pathological history of this affection. They reinforce the conclusion that the susceptibility of children is greater than that of adults, and also strengthen the view that the cause of the disease, toxic though it probably is, does not necessarily stand in a direct relation to an obvious infectious malady.

Freyhan's[7] case is as follows: The patient was a boy of fifteen, and of good health, except for a tuberculous inflammation of the middle ear of long standing, which could not be considered as the cause of the subsequent illness, especially as the cerebro spinal fluid obtained by lumbar puncture was free from tubercular organisms. After a moderate attack of influenza the patient showed symptoms of cerebral disorder (transitory hemiplegia and apathy); and to these succeeded paresis of the right side of the face (of the peripheral type) and of the associated eye muscles; and, later, ataxia, vertigo, vomitting,and other symptoms. There was no fever at any time throughout the illness, and complete recovery occurred in the course of four months.

Nauwerck[8] reported the following cases with post mortem examination: The first is that of a girl fourteen years old, who was seized, during the influenza epidemic, but without having herself shown clear signs of infection, with fever which remitted and again increased, left-sided convulsions, vomiting, left hemiplegia. Consciousness became clouded quite early. The eyes were not affected. After a fortnight from the beginning of the illness, toward the end of which symptoms had occurred, restlessness and convulsions set in, with loss of consciousness. Death occurred the next day. At the post-mortem examination the cerebral substance was found soft, and numerous foci of hæmorrhagic softening were discovered in various parts of both hemispheres. No bacteria could be found by the usual methods.

The second is that of a girl of nineteen, who had been in excellent health except for a trifling cold in the head and pains in the limbs. The cerebral attack began sud denly, with headache, uncertainty of gait, faintness, vom iting, loss of pupillary light reflex and slow pulse. That same night the pulse became still slower, fifty beats to the minute. The patient became dull and restless and died on the following day. At the post-mortem examination a hæmorrhagic focus of the size of a walnut was found in the right lobe of the cerebellum. This was surrounded by a zone of inflammatory softening; and bacteria, believed to be identical with the influenza bacillus, were found here, and also in the ventricular fluid, which showed signs of being of inflammatory origin. Nauwerck believes, with Leichtenstern, that these hæmorrhagic foci may sometimes be of embolic (bacterial) origin; and, with Pfuhl and many others, that the symptoms on the part of the nervous system may be the first sign of the infection.

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[7] *Deutsche Med. Wochenschr.*, 1895, Vol. 7, p. 643.

[8] *Deutsche Med. Wochenschr.*, 1895, Vol. 7, p. 393.

Brie (Duren)[9] likewise reports a fatal case of unknown origin, with autopsy, the patient being, this time, a woman of thirty-six. The duration of the illness was nine days. The symptoms were loss of appetite, abdominal pains, trifling fever, headaches with increasing hebetude; later, rigidity of the neck muscles, dilatation of one pupil, coma, rapid increase of temperature up to the time of death. The post mortem examination showed the spinal cord, medulla oblongata, pons and cerebellum to be free from obvious lesions, and this observation increases the probability that the rigidity of the neck muscles observed in such cases is not especially a sign of meningitis. The cerebral hemispheres showed signs of general pressure—dryness of the surface, and flattening of the convolution—and within their substance, on both sides, numerous foci of hæmorrhagic softening small and large, were found.

Finally, a brief reference to two other important papers, bearing indirectly upon this subject, may be in place. The first is that of Kaiser,[10] who discusses at length a fatal case in which the bulbar lesions of Wernicke's disease were associated with similar changes involving the nuclei of the spinal cord. The case is especially noteworthy for the present discussion on the following grounds:

1. The patient was a young and healthy man, not alcoholic and not sick with any infectious disease so far as known.

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[9] *Allg. Zeitschr. f. Psych.* Bd. 53. p. 604.

[10] Zur Kenntniss der Poliencephalomyelitis acuta. *Deutsche Zeitschr. für Nervenheilkunde*, 1895, Vol. 7, p 359.

In this respect the case deserves to be ranked with those of the typical poliomyelitis of children and adults, and although the belief is growing that this latter disease is really of infectious origin, a belief strengthened by the occurrence of epidemics such as that recently described by Caverly,[11] in which cases of somewhat different character are apt to occur, side by side, yet it must be remembered that we have as yet no idea what the infectious agent is. In view of our ignorance on this point we cannot properly deny that on this unknown agent may be the effective causes, perhaps as a secondary infection, in a portion of the cases which we are inclined to attribute to some prevalent disease of a recognized sort.

2. The author discusses at some length the symptom of drowsiness, which may be present in a striking form before in the early stages of the illness, and shows that it presents itself not infrequently in these cases where eye paralysis is prominent, and seems to be associated with an involvement of the walls of the third ventricle. In the case observed by Dr. Drake and myself this somnolence seems to have been well marked for many days.

The second paper to which I would refer is that by Dinkler,[12] in which a case is described where vascular lesions, analogous to those described, occurred, again in a young child, without obvious cause except a severe blow on the head two and a half years before. In view of the length of this interval, no importance would have been attributed to this influence but that the child had been in an abnormal state ever since the injury, In view of this case, taken in conjunction with so many others where injuries have counted as partial causes of complex disorders of later occurrence, the possibility cannot be excluded that the mysterious susceptibility of the highly vascular nuclear gray matter was heightened by the fall on the head in this case, the more so that a general congestion of the cerebral cortex was found at the post-mortem examination.

The possibility that some toxic agent was also at work cannot be set aside, but none seems to have been apparent.

The case observed by myself is as follows:

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[11] *Jr. of Amer. Med. Ass.* January 4. 1896.

[12] Mittheilung eines letal verlaufenden Falles von traumatischer Gehirnerkrankung mit dem anatomischen Befunde einer Poliencephalitis hæmorrh. acuta. *Deutsche Zeitschr. f. Nervenheilkunde*, 1895, Vol. 7, p. 465.

The patient was a healthy boy, nearly thirteen years of age, whom I saw in consultation with Dr. E. T. Drake, of Franklin, N. H., who has kindly given me his notes of the case to supplement my own.

In November, 1895, he passed through a moderate attack of mumps, and had apparently been entirely well for ten days before the present illness began. On December 28, 1895, he woke up feeling poorly, and went into his brother's room saying that he thought he had "a squint in one eye," in spite of which, however, he went out of doors and followed his usual occupations. The next morning he had a partial ptosis of both eyelids, especially the right, and impairment of motion of the right eye, which is said to have turned inward. He also became slightly deaf at this time, though at the moment not much was thought of it. He also complained of pain in the forehead, which developed into an intense and persistent headache, and of dullness and drowsiness which soon became so marked that he could be aroused only with difficulty, though it could be done. Two days later he was seen by Dr. Drake, who found his temperature to be 100° F. By this time the paralysis of the eyes, and the deafness had increased, and for the next week all his symptoms grew steadily worse. At times he would be slightly delirious, but generally his consciousness was fairly good after he had been fairly aroused. Sensations of numbness were complained of, referred to the left arm and leg, and these limbs were both weaker than those of the right side, though there was no paralysis. In fact, he had to be restrained by force from getting out of bed and could stand erect on both feet. The temperature ranged from 99° to 100°; the pulse from 70 to 90.

I saw him on January 1, 1896. He was then lying in bed, picking restlessly at the bed clothes like a typhoid patient, but using the right hand more than the left. His nose was filled up with mucus which, apparently, he had not strength enough to expel. His deafness seemed almost complete. Both eyes were motionless and fixed in the median line. The pupils were rather small and did not respond to light. There was ptosis of both lids. The power of swallowing was considerably impaired. Thus, milk would run out from his lips, and the reason seemed to be because the latter could not be pressed firmly together. It was, indeed, noticeable that there was paresis of the lower facial muscles, especially on the left side.

An ophthalmoscopic examination showed double optic neuritis of moderate intensity. He could not be made to respond to questions; resisted attempts to instil cocaine into the eye, and gave indications of pain when pricked. The face was somewhat flushed and rather spotty; the ears rather red. The knee jerks and wrist jerks were absent on both sides. The plantar reflexes were present, but the right was greater than the left. The abdominal reflexes were present. So far as could be judged by the motions of the patient, the sensibility of the skin over the whole left leg, the left side of the body, and the left arm was noticeably less than that of the corresponding parts of the right side, but no difference could be made out with certainty between the two sides of the face.

The treatment followed was mainly expectant, care being taken to get in a good quantity of nourishment and full doses of strychnia. The case at that time looked very serious, but with the remembrance of Oppenheim's reports in my mind, I ventured to hold out the possibility of a favorable outcome, at least as regards life. Since then I have learned through three letters and one interview with Dr. Drake that this hope has been more than justified. On January 4th, he was reported as free from fever, but with total loss of hearing and almost complete loss of eyesight, and the pupils were more contracted than when I saw him and did not respond to light. The right eye could be moved to a slight degree. On January 8th, he showed more consciousness and recognized Dr. Drake at his visit. He could raise the lids a little and apparently heard somewhat better. January 30th, Dr. Drake wrote, "Our boy is doing finely. His hearing is nearly, if not quite normal. He seems to have perfect control of the left eye and lid, and the sight is nearly normal. He can open the right eye only about two-thirds, and the ball turns out, and the sight is not yet normal. When I ask him to fix his eyes upon my finger at his left, lateral nystagmus is noticeable (in right eye only). When he uses both eyes he sees double and the right object is the more distinct. The appetite is good and he sleeps well. His speech is quite natural and mind clear. Memory is good. Fainted about a week since while sitting up in bed drinking milk. Sits up half an hour a day. The boy is anxious to get about the house."

On March 18th, that is, about three months after the onset of the illness, the boy was brought to see me with his mother on their way to the South, and I obtained the following additional facts:

His eyesight was still somewhat defective, though improving. Double vision was still present and tests showed that the vision of the right eye was slightly deficient and that of the left normal. The motion of the right eye was also still imperfect, though not wholly lost for either direction. That for the left eye was good. The pupils on both sides failed to respond to light except very slightly. The left was of moderate size, slightly irregular in outline; the right somewhat smaller than the left. Traces of optic neuritis were still noticeable on both sides. The hearing was thought by the patient to be as good as ever, but tests with the watch showed that his hearing distance was but five inches on the left side as compared with five feet on the right. The bone conduction was about equal for the two sides. The grasp of the right hand was somewhat weaker than that of the left. The knee jerk was lacking for the right side and normal for the left. He looked strong and well and his mental condition was excellent. His weight was fifteen pounds greater than before his illness. In spite of this, on the whole favorable, condition, he had shown one symptom, for the outcome of which I shall watch with interest. Ever since his illness, namely, he had had epileptiform attacks, which at first recurred many times a day, sometimes he would even have several attacks in the course of a few minutes. They last from five to fifteen seconds, and during that period there is twitching of the right arm and the right side of the face. He is unable to speak, or talks gibberish, and is evidently dazed though he does not wholly lose consciousness. Of late these attacks had been less frequent.

A recent letter from Dr. Drake, of the date of November 16th, 1896, describes the present condition as follows: "The diplopia is still present, but he is learning to ignore the left and fainter image, so that he can read and study without much annoyance. As regards his hearing, he can hear the tick of a watch with the right ear at thirty inches, with the left at twenty inches. He is entirely free from the epileptiform seizures, but my impression is that he is not so strong mentally as before. A sister says, 'he does many queer things; is destructive; is not irritable nor quick to take offence, but does such funny things.'"

The remarkable number, character and variety of these symptoms, involving epileptiform seizures, hemiparesis, and coma, ophthalmoplegia, temporary loss of hearing, and impairment of deglutition and loss of knee jerks surely points to wide-spread lesions, reaching from the cortex to the medulla, and the general resemblance of the symptom-complex to that present in the cases described and collected by Oppenheim justified, at least provisionally, the diagnosis of disseminated encephalitis. The extreme loss of hearing seems to be an exceptional but not a wholly new feature. Some degree of deafness was present in one of the cases cited, tinnitus in another, and word deafness in a third. From the early occurrence of the symptom and its association with the ophthalmoplegia, I am inclined to refer it to an affection of the auditory nerves or centres, rare as this lesion, for some reason, seems to be. The relation of this case to the mumps must be left in question.

It is evident that the symptoms in different cases of this affection must differ a great deal in view of the variability of the seat of the lesion and the nature of the influence which causes it. There are, however, certain clinical facts which it is important to bear in mind: (1) For some reason, a large proportion of the patients (omitting the alcoholic cases) have been children. (2) The temperature, although usually high, is occasionally but little elevated and may be even below the normal. This is said to occur particularly often with the alcoholic poliencephalitis of Wernicke, but the same fact has been observed with cases of other sorts, so that the alcohol is probably not itself the cause of the lowered temperature. (3) Even when the encephalitis follows an acute disease, a period of one to several weeks may pass before the outbreak. This leads naturally to a closer consideration of what the real cause of the disease may be. The principle antecedents, beside the influenza, have been alcohol, sulphurous acid, fright, trauma, puerperal disease, cerebro spinal meningitis, ulcerative endocarditis, but sometimes no morbid antecedents could be discovered. Oppenheim raises the question whether a light attack of influenza which had been recovered from some weeks before could have had to do with the onset of the affection in one case, and thought that this was not impossible. The case which I reported in 1892 was that of a man in early middle life, who had had in January a well-marked attack of influenza, during which intense headache was a prominent symptom. From this he apparently recovered entirely, so that he had returned to work, though still troubled more or less with headache, when, some eight weeks after the occurrence of the primary seizure, the cerebral symptoms came on. The actual mode of onset was quite interesting. The patient was seized with a violent fit of sneezing while at his work, and before this was over, he found that he could neither hear nor see. The hearing quickly returned, and the vision almost entirely, but not wholly. From that time on he was confined to the bed with intense headache and delirium. Two weeks later he had an attack of left hemiplegia and slight impairment of speech. The hemiplegia began to lessen after two days and at the end of a week had practically disappeared. At the time that I saw him, which was five weeks after the beginning of his illness, although he was slowly mending, I found double optic neuritis with swelling and hæmorrhages. In the end, however, he recovered.

Fürbringer speaks of a case of encephalitis following scarlet fever where the cerebral symptoms showed themselves in the fifth week after the beginning of the primary illness.

In all these cases it is, of course, quite possible that we may have to deal with a secondary infection, or with some unappreciated cause, but it is also true that, on the one hand, the bacterial toxines may linger in the tissues far longer than one would conceive to be probable, and, on the other hand, that the outbreak which eventually comes may have been for some time closer to the surface than we had supposed. A large number of facts could, for example, be cited to show that in many cases of toxic neuritis due to the chronic influence of some poison, the final outbreak had long been prepared for by specific nutritive changes, some of which would probably have been recognizable under the microscope, just as in Gombault's lead poisoned guinea pigs signs of degenerative neuritis were discovered, though no paralysis had been present.

In this connection I wish to refer to a case (not hitherto reported) of acute ataxia of all four extremities, of sudden onset and ending in partial recovery, the patient having been a healthy man in middle life, an intelligent mechanic. The case was evidently one of the kind which has been described by Leyden as *acute ataxia*, and I mention it partly because these cases are probably due to an acute disseminated myelitis, analogous to the disseminated encephalitis, but sometimes terminating in sclerosis; partly in order to call attention to the fact that this patient was taken ill during a severe epidemic of influenza, though without having shown any of the catarrhal symptoms. Of course it is impossible to assert with confidence that the influenza poison was responsible for his final illness, but there are indications that just as diphtheritic paralysis may occur where little or no recognizable sore throat has gone before, so, also, something similar may be true for the case of influenza. (4) Must we assume that in cases presenting the clinical aspect of toxic encephalitis all the symptoms are referable to recognizable lesions, even such trifling ones as those recorded in some of the post-mortem reports above referred to?

It seems to me that this question is distinctly to be answered in the negative. Chemical changes in nerve-cells, of toxic origin—using that term in its widest sense—are fully capable of leading to abnormal activity or to paralysis, and even when gross lesions are found, their importance often consists as much in affording a sign that the conditions necessary for toxic influence were present, as in defining the actual cause of the symptoms. Disordered vaso motor action is another partial cause.

It seems to be certain that some of the irritative symptoms usually considered indicative of inflammation of the cerebral membranes, in this class of cases, such as spasm of the muscles of the neck and back, may occur without this lesion;[1] and even such paralyses as follow diptheria may be independent of any gross change that we can as yet discover. I would call attention in this connection to two cases of personal observation. The first is that of an elderly lady recorded in the paper already referred to,[2] who, during a convalescence from influenza, suffered from a series of frequently recurring typical Jacksonian convulsions, at first unattended with loss of consciousness, but ending on the third day in hemiplegia and death, where the autopsy and microscopic examination revealed nothing but the signs (as I finally concluded) of universal œdema cerebri. The second is a case of a kind to which Dr. Wm. Osler referred in a discussion as to the nature of "typhoid spine" in the American Journal of Medical Sciences for 1894, (vol. CVII., p. 23). The patient was a middle aged man, seen in consultation with Dr. Hodgdon, of Arlington, who, during a protracted convalescence from a severe attack of typhoid, suffered extremely from violent and painful spasm of the extensor muscles of the back, brought on by the slightest movement, and suggesting the diagnosis of meningitis, which was, however, quickly abandoned. Eventually, while sitting up to take some food, he was seized with an attack of vomiting and instantly died. An autopsy, made by Dr. W. F. Whitney, showed a high degree of anæmia throughout the spinal cord, medulla oblongata and membranes, but no trace of gross lesion.

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[1] In the discussion of this paper Dr. Osler especially called attention to this fact.

[2] On the Nervous Complications of Influenza. Boston Medical and Surgical Journal, October 6th, 1892.